ANNALS OF HUMAN BIOLOGY, 1982, VOL. 9, NO. 3, 253–263

Evidence for directional non-additivity in the genetics of finger ridge counts

N. G. MARTIN, D. Z. LOESCH and R. JARDINE

Department of Population Biology, Research School of Biological Sciences, Australian National University, Canberra

and H. S. BERRY

Department of Genetics, University of Birmingham

Received 13 November 1981

Summary. A genetical analysis of variation in finger ridge counts of 221 pairs of twins and 80 pairs of opposite sex siblings has been carried out. Negative regression of DZ and sibling pair variances on pair means suggests the action of non-additive genes or unequal gene frequencies tending to increase finger ridge counts. Negative skewness of the distributions supports this view. While models including dominance or epistasis are not a significant improvement over purely additive genetic models, it is regarded as important that large and positive values of non-additive genetic variance are estimated. The evolutionary importance of dominance and epistasis for greater finger ridge counts is discussed.

1. Introduction

Since the extensive work of Holt, finger ridge counts and particularly the total ridge count (TRC) have been considered classical examples of traits under perfectly additive polygenic inheritance.

It has become evident, however, that this model is not in complete agreement with the data. Holt (1955, 1968) pointed out the negative skewness of the distribution of TRC, but she did not consider this incompatible with the hypothesis of additivity. Furthermore, the values of correlation coefficients between relatives for total ridge count as well as those for counts on individual fingers slightly, although not consistently, deviate from theoretical expectations based on the assumption of perfectly additive inheritance (see, for example, Holt 1956, 1957, 1968, Vogelius-Andersen 1963, Bocheńska 1964, Loesch 1971, Matsuda and Matsunaga 1971, Jeliesiejew and Marcinkiewicz 1972, Mi and Rashad 1975). Spence *et al.* (1977) used the sensitive technique of pedigree analysis to re-examine Holt's family data and found evidence for dominance variance for TRC. Other evidence comes from inter-racial crosses. Hybrids between Australian Aboriginals and Europeans have mean TRC values significantly greater than the midparent, suggesting dominance in the direction of higher ridge counts (Robson and Parsons 1967, Singh 1979).

In the present study a genetic model-fitting approach has been applied to twin and sibling data in order to define more precisely the components contributing to the total variance of ridge counts on individual fingers and their sum (TRC). The same twin data have been analysed elsewhere by Loesch (1979) using methods which were not suitable for detecting dominance.

2. The data

The study is based on finger prints from 221 paris of like-sexed twins collected in schools in the city of Wrocław, Poland. There are 60 male and 50 female monozygous

0301 4460/82/0903 0253 $02·00

(MZ) twin pairs and 62 male and 49 female dizygous (DZ) twin pairs. An additional 80 pairs of opposite-sex full-siblings (FSO) of Polish origin from the Olkusz region in Silesia have also been included.

Zygosity determination of the twins was essentially based on testing blood groups: ABO, MN, Rh (tests with anti-C, -c, -D, -E, -e) and other genetic markers (Hp, Gc, Gm^a). In addition, 74 twin pairs were tested for Gm (b, f, 1, 2), PGM, GPT, AK, Inv, 6PGD and ACP.

In the rare cases in which zygosity could not be satisfactorily diagnosed on the basis of genetic markers, a polysymptomatic test of similarity was used (Verschuer 1928, Siemens 1937) and a probability of MZ/DZ zygosity was estimated according to the method described by Wyslouchowa and Orczykowska-Światkowska (1969).

The characters investigated were ridge counts on individual fingers (the highest count being taken where there was more than one count to a pattern) and total ridge count (TRC), which is the sum of the individual counts on all ten fingers (see Holt 1968).

The means and standard deviations for the characters are given in table 1. Because twins are not genetically independent observations, the appropriate tests of significance are not obvious. It is clear, however, that, consistent with what is commonly known, male counts are generally higher than female and that the counts on the right thumb are greater than on the left. In the same table the skewness (g_1) of the distributions of ridge counts is also given. Nearly all of these coefficients are significantly negative and the interpretation of this will be discussed later.

Table 1. Means, standard deviations and skewness (g_1) of finger ridge counts separately for males and females.

	Males			Females		
Variable†	Mean	Standard Deviation	Skewness	Mean	Standard Deviation	Skewness
L1	17·00	6·08	−0·49***	15·32	5·91	−0·83***
L2	12·33	6·13	−0·38**	11·48	6·59	−0·41**
L3	13·84	5·66	−0·44**	12·71	6·15	−0·64***
L4	17·76	5·16	−0·45**	16·10	6·28	−0·94***
L5	14·30	4·49	−0·59***	12·22	5·11	−0·58***
R1	19·54	5·52	−0·77***	18·07	5·41	−1·06***
R2	13·05	7·07	−0·13	11·99	7·51	−0·35*
R3	12·74	5·95	0·01	12·63	5·72	−0·33*
R4	17·66	5·36	−0·79***	16·27	5·73	−0·68***
R5	14·32	4·67	−0·44**	12·34	5·64	−0·27
TRC	152·54	43·37	−0·31*	139·12	48·55	−0·70***

† Finger 1 is the thumb, finger 5 the little finger.
* $0{\cdot}01 < P < 0{\cdot}05$; ** $0{\cdot}001 < P < 0{\cdot}01$; *** $P < 0{\cdot}001$.

3. Methods and results

Detecting directional non-additivity

It has been shown that the probability of detecting dominance by fitting models to twin mean squares is low, even under ideal conditions of high heritability and complete dominance (Martin *et al.* 1978). At the same time, however, the twin design provides another quite powerful test for detecting genetic non-additivity if it has a strong directional component.

Jinks and Fulker (1970) showed that regressing MZ pair variances on MZ pair means is a test for $G \times E_1$ interaction and Martin and Eysenk (1976) found that this test was capable of detecting $G \times E$ accounting for only 1% of the total variance with only 95 pairs of MZ twins. Martin *et al.* (1978) showed that if the same test were applied to DZ twins (or sibs), it would detect terms in $-d^2_{ah_a}$, where d_a is the additive deviation at a locus A and h_a is the dominance deviation at the same locus. As Fisher, Immer and Tedin (1932) originally showed, the coefficient of skewness (g_1) detects the same terms. Thus, if the MZ data show no indication of $G \times E_1$ interaction, but there is either a negative regression of DZ pair variances on pair sums or a negative coefficient of skewness, or both, then one might infer that there is dominance acting predominantly in the increasing direction. Positive regression or skewness would indicate dominance acting in the decreasing direction. The same effects will be observed if the average frequencies of increasing and decreasing alleles at loci affecting the trait are unequal (Martin *et al.* 1978). If the increasing allele is more common, negative regressions and skewness will result.

Linear mean-variance regression coefficients for individual finger and total ridge counts are shown in table 2. Only one in twenty of the individual finger MZ regression coefficients is significant at the 5% level. Out of 30 of the DZ and sibling regression coefficients for individual fingers, 12 are significantly negative, at least at the 5% level; however, the three regression coefficients for TRC, although negative, are not significant. Mean-variance regressions appear to be most consistent for L4 and least for R3. This may reflect genuine variation in the degree of dominance from finger to finger, but it is more probably a consequence of the less than perfect power of the regression test. In support of this, we note that all but one of the 30 regression coefficients are negative and large in comparison with their MZ counterparts. Thus we can infer that there is no $G \times E_1$ interaction but that genetic non-additivity or unequal gene frequencies are influencing the individual counts.

The skewness of the individual finger distributions in males and females supports this conclusion; all but three in twenty show significant negative skewness (table 1).

Fitting models to the mean squares

In spite of the theoretically low chance of detecting genetic non-additivity by fitting models to mean squares, we shall do so here because some evidence for it has already been obtained from the regression analysis. The procedure is described extensively

Table 2. Tests for directional non-additivity: linear mean-variance regression coefficients.

	MZ		DZ		
Variable	Males	Females	Males	Females	Opposite sex sibs
LI	−0·09	−0·87	−1·31	−2·26	−4·16*
L2	−0·02	−0·54	−2·64*	−1·87	−2·88
L3	−0·61	−0·24	−0·70	−2·68	−2·25
L4	0·14	−0·63	−2·14**	−4·38**	−4·68**
L5	0·00	−0·13	−2·04**	−0·43	−1·71
R1	−0·33	−0·60	−0·77	−5·76***	−4·01**
R2	−0·09	−0·09	−0·85	−3·47*	0·30
R3	−0·36	−0·30	−0·46	−1·25	−1·55
R4	0·42*	−0·58	−3·21***	−1·05	−1·85
R5	−0·09	−0·08	−0·78*	−0·96	−1·82
TRC	−0·44	−0·62	−5·73	−12·16	−11·11

elsewhere (e.g. Martin 1975, Eaves and Eysenck 1975). Firstly, between- and within-pairs mean squares are calculated for each of the five twin or sibling groups. These are shown for each individual finger count and for TRC in table 3. Models are then fitted to the mean squares by the procedure of iterative weighted least squares. Computation of the expected mean squares from the least-squares parameter estimates provides a chi-square test of goodness-of-fit of the model.

The full model for mean squares from a twin study is shown in table 4. The expectations for sibs reared together are the same as for DZ twins. This model includes parameters for individual environmental variance (E_1), shared family environment (E_2), additive genetic variance (D_R) and dominance (H_R) (Mather and Jinks 1971). It should be noted, however, that the expectations for H_R and for additive × additive epistasis (I_R) are identical in MZ and DZ twins (Mather 1974), so we must remain agnostic about the true nature of any non-additive genetic variation detected. We adopt the parsimonious approach of only fitting a more complex model when a simpler one has failed. The results of all model-fitting are shown in table 5, but by way of illustration, we shall consider the results for TRC in some detail.

Environmental models comprising only E_1, or E_1 and E_2 fail badly, but a model including only E_1 and D_R gives an excellent account of the data in both sexes and in the data jointly. We can calculate a chi-square for the heterogeneity of fit over sexes by adding the χ^2_2 values for males and females and subtracting from the χ^2_6 of the joint data. This value ($\chi^2_2 = 3{\cdot}75$) is not significant. We are thus entitled to fit the model to the full data set including the opposite-sex siblings and this gives a good fit to the data ($\chi^2_8 = 7{\cdot}35$). We can calculate a heritability

$$\hat{h}^2 = \frac{\frac{1}{2}\hat{D}_R}{\frac{1}{2}\hat{D}_R + \hat{E}_1} = 0{\cdot}95 \pm 0{\cdot}01.$$

There is no need to fit further parameters since we already have an adequate account of the data. However, to see whether there is any suggestion of dominance, a model

Table 3. Observed mean squares for finger ridge counts.

		d.f.	L1	L2	L3	L4	L5	R1	R2	R3	R4	R5	TRC
MZM	MSb	59	76·6	65·6	55·8	38·8	28·5	60·2	91·1	61·4	45·8	35·7	3468
	MSw	60	5·3	6·5	5·6	4·3	4·0	6·5	11·8	13·5	3·6	4·5	113
MZF	MSb	49	58·9	74·4	68·3	72·2	52·0	55·8	100·6	56·2	61·9	61·9	4824
	MSw	50	9·0	17·9	6·3	9·1	2·1	7·7	11·5	6·7	5·8	5·2	84
DZM	MSb	61	42·0	42·8	47·1	46·0	32·5	39·2	60·7	46·3	43·6	36·1	2867
	MSw	62	25·3	35·0	20·1	17·9	15·8	17·1	38·1	21·4	20·7	11·3	1139
DZF	MSb	48	50·5	49·0	52·2	44·1	38·3	32·4	67·7	46·4	40·2	35·2	3064
	MSw	49	22·3	32·8	25·8	33·5	13·3	20·4	47·4	22·5	24·2	25·7	1533
FSO	MSb	79	37·9	53·8	61·0	46·0	34·9	47·1	66·1	44·8	38·8	28·5	2658
	MSw	79	22·9	34·7	23·9	21·2	18·5	18·5	39·8	28·7	16·2	13·8	1096

Table 4. Basic model for mean squares of twins reared together.

	E_1	E_2	D_R	H_R/I_R
MZ between	1	2	1	$\frac{1}{2}$
within	1	0	0	0
DZ between	1	2	$\frac{3}{4}$	$\frac{5}{16}$
within	1	0	$\frac{1}{4}$	$\frac{3}{16}$

Table 5. Results of fitting models to the raw data.

Variable	Model	Data set	$\hat{E}_1$	$\hat{D}_R$	$\hat{H}_R/\hat{I}_R$	χ^2†	$\hat{h}^2$
L1	E_1D_R	M	5·49***	65·00***	—	3·04	0·86±0·03
		F	9·07***	52·58***	—	0·09	0·74±0·06
		8	7·15***	56·17***	—	6·52	0·81±0·03
		10	7·30***	55·84***	—	9·25	0·79±0·03
	$E_1D_RH_R$	M	5·27***	15·09	94·38	0·85	
		F	9·09***	54·38	−3·53	0·09	
		8	6·97***	34·01	48·32	5·73	
		10	6·92***	26·54	56·58	7·39	
L2	E_1D_R	M	7·22***	67·13***	—	9·25*	0·82±0·04
		F	18·22***	50·69***	—	0·66	0·58±0·08
		8	12·75***	57·05***	—	17·30**	0·69±0·04
		10	13·40***	58·16***	—	18·54*	0·68±0·04
	$E_1D_RH_R$	M	6·54***	−33·16	191·73**	0·13	
		F	17·56***	20·94	60·83	0·30	
		8	11·66***	−10·20	134·36*	13·57*	
		10	11·73***	−1·30	121·23**	14·06	
L3	E_1D_R	M	5·71***	53·93***	—	0·22	0·83±0·04
		F	6·52***	65·28***	—	0·58	0·83±0·04
		8	6·08***	58·97***	—	2·47	0·83±0·03
		10	6·10***	63·10***	—	3·88	0·84±0·02
	$E_1D_RH_R$	M	5·67***	47·91*	11·52	0·17	
		F	6·37***	38·23	51·53	0·04	
		8	5·99***	43·82*	28·93	1·89	
		10	6·02***	54·14**	17·01	3·65	
L4	E_1D_R	M	4·44***	47·07***	—	2·92	0·84±0·03
		F	9·95***	62·83***	—	3·70	0·76±0·05
		8	6·89***	54·23***	—	22·33**	0·79±0·03
		10	6·93***	54·05***	—	22·35**	0·80±0·03
	$E_1D_RH_R$	M	4·42***	42·53*	8·68	2·91	
		F	9·13***	−16·71	155·31*	0·04	
		8	6·60***	20·63	64·97	18·24**	
		10	6·60***	30·33*	46·02	19·32**	
L5	E_1D_R	F	4·34***	34·31***	—	3·89	0·80±0·04
		M	2·15***	47·80***	—	0·12	0·92±0·02
		8	3·26***	40·88***	—	10·54	0·86±0·02
		10	3·35***	43·51***	—	13·75	0·87±0·02
	$E_1D_RH_R$	M	4·19***	19·09	29·58	3·04	
		F	2·16***	54·91*	−13·13	0·04	
		8	3·23***	33·13*	14·64	10·41	
		10	3·24***	26·50*	31·85	11·81	
R1	E_1D_R	M	6·42***	47·53***	—	0·67	0·79±0·04
		F	7·91***	42·66***	—	1·39	0·73±0·06
		8	7·07***	45·47***	—	2·73	0·76±0·04
		10	7·03***	47·11***	—	3·22	0·77±0·03
	$E_1D_RH_R$	M	6·47***	53·13*	−10·79	0·63	
		F	7·59***	13·83	56·58	0·65	
		8	6·98***	36·09*	18·22	2·55	
		10	7·02***	46·53**	1·13	3·22	

Table 5. (*continued*)

Variable	Model	Data set	$\hat{E}_1$	$\hat{D}_R$	$\hat{H}_R/\hat{I}_R$	χ^2†	$\hat{h}^2$
R2	E_1D_R	M	12·45***	78·38***	—	1·87	0·76±0·05
		F	12·49***	94·29***	—	3·48	0·79±0·05
		8	12·49***	85·35***	—	6·46	0·77±0·03
		10	12·92***	84·33***	—	8·29	0·77±0·03
	$E_1D_RH_R$	M	11·77***	16·13	121·63	0·04	
		F	11·59***	−11·17	203·97*	0·01	
		8	11·69***	3·81	158·57	0·96	
		10	11·69***	11·88	142·13*	1·10	
R3	E_1D_R	M	12·94***	44·56***	—	0·59	0·63±0·07
		F	6·88***	53·58***	—	0·44	0·80±0·05
		8	10·36***	48·01***	—	6·13	0·70±0·04
		10	10·85***	49·06***	—	8·26	0·69±0·04
	$E_1D_RH_R$	M	13·34***	64·10*	−39·56	0·25	
		F	6·74***	36·82	32·40	0·15	
		8	10·41***	51·41	−6·77	6·05	
		10	10·45***	34·22*	30·11	7·75	
R4	E_1D_R	M	3·83***	52·86***	—	2·55	0·87±0·03
		F	6·09***	56·09***	—	1·74	0·82±0·04
		8	4·86***	54·21***	—	7·66	0·85±0·02
		10	4·85***	51·44***	—	9·17	0·84±0·02
	$E_1D_RH_R$	M	3·72***	26·91	48·76	1·32	
		F	5·82***	12·12	84·05	0·05	
		8	4·69***	21·29	62·41	4·85	
		10	4·68***	31·69*	37·41	7·06	
R5	E_1D_R	M	4·46***	34·53***	—	1·91	0·79±0·04
		F	5·56***	56·19***	—	4·13	0·83±0·04
		8	5·00***	43·92***	—	16·09*	0·81±0·03
		10	5·00***	40·54***	—	20·61**	0·80±0·03
	$E_1D_RH_R$	M	4·65***	54·49**	−38·74	0·55	
		F	5·21***	−11·88	129·56*	0·17	
		8	4·89***	28·09	30·37	13·40*	
		10	4·86***	28·93*	22·37	18·15*	
TRC	E_1D_R	M	114·84***	3692·26***	—	0·54	0·94±0·01
		F	84·78***	4812·53***	—	1·59	0·97±0·01
		8	101·23***	4189·91***	—	5·88	0·95±0·01
		10	101·46***	4024·63***	—	7·35	0·95±0·01
	$E_1D_RH_R$	M	114·12***	2835·35*	1568·78	0·24	
		F	84·02***	1864·10	5360·83	0·07	
		8	100·36***	2443·70*	3184·32	3·95	
		10	100·27***	2634·81**	2503·06	5·23	

† Degrees of freedom for χ^2 are $n-k$, where there are n statistics (4 for sexes separately, 8 jointly, 10 including opposite sex sibs) and k parameters estimated.

including E_1, D_R and H_R has been fitted. This does not result in a significant reduction in chi-square, nor are any of the estimates of H_R significant. When this model is fitted, it can be shown that $\hat{H}_R = H_R - 8E_2$, so if there is any E_2 present a negative value for $\hat{H}_R$ will usually be obtained (Martin *et al.* 1978). Since nearly every trait is influenced by some E_2 effects, it is unusual to obtain a large positive value for $\hat{H}_R$. The same authors

also showed that even in the extreme case of a character with 90% broad heritability, complete dominance and no E_2, 3330 pairs (half MZ, half DZ) would be required to reject an $E_1 D_R$ model at the 5% level in 95% of such analyses. Considering the low power provided by the data, it is noteworthy that we have even obtained large positive, albeit non-significant, estimates of H_R. Clearly this would not occur if there were any shared intra-uterine environmental influences (E_2) on TRC. Broad and narrow heritabilities based on the $E_1 D_R H_R$ model can now be calculated as

$$\hat{h}^2_N = \frac{\frac{1}{2}\hat{D}_R}{\frac{1}{2}\hat{D}_R + \frac{1}{4}\hat{H}_R + \hat{E}_1} = 0{\cdot}64 \pm 0{\cdot}28$$

and

$$\hat{h}^2_B = \frac{\frac{1}{2}\hat{D}_R + \frac{1}{4}\hat{H}_R}{\frac{1}{2}\hat{D}_R + \frac{1}{4}\hat{H}_R + \hat{E}_1} = 0{\cdot}95 \pm 0{\cdot}01.$$

Although there is no significant heterogeneity of fit of the $E_1 D_R$ model over sexes, a model is shown in table 6 which takes account of such heterogeneity and allows inclusion of the opposite-sex sibling data (Eaves 1977, Clark *et al.* 1980, 1981 a, b). Separate E_1 and D_R terms are fitted for males and females and an additional term, D_{Rmf}, estimates the covariance of additive gene action in males and females. When fitted to the TRC data this model yields estimates of:

$$\hat{E}_{1f} = \ \ 85^{***}$$
$$\hat{E}_{1m} = \ 115^{***}$$
$$\hat{D}_{Rf} = 4539^{***}$$
$$\hat{D}_{Rm} = 3551^{***}$$
$$\hat{D}_{Rmf} = 3864^{***}$$

and the goodness-of-fit is indicated by $\chi^2_5 = 3{\cdot}60$. The heritabilities in the two sexes are $h^2_f = 0{\cdot}96 \pm 0{\cdot}01$ and $h^2_m = 0{\cdot}94 \pm 0{\cdot}01$. The correlation between additive gene action in the two sexes can be estimated as:

$$r_{D_{Rmf}} = \frac{\hat{D}_{Rmf}}{\sqrt{\hat{D}_{Rm}\hat{D}_{Rf}}} = 0{\cdot}96.$$

Table 6. Model for different environmental and genetic effects in males and females.

Mean square		E_{1m}	E_{1f}	D_{Rm}	D_{Rf}	D_{Rmf}
MZM	between	1	0	1	0	0
	within	1	0	0	0	0
MZF	between	0	1	0	1	0
	within	0	1	0	0	0
DZM	between	1	0	$\frac{3}{4}$	0	0
	within	1	0	$\frac{1}{4}$	0	0
DZF	between	0	1	0	$\frac{3}{4}$	0
	within	0	1	0	$\frac{1}{4}$	0
FSO	between	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{4}$	$\frac{1}{4}$	$\frac{1}{4}$
	within	$\frac{1}{2}$	$\frac{1}{2}$	$\frac{1}{4}$	$\frac{1}{4}$	$-\frac{1}{4}$

$D_{Rm} = D_R$ effect for males
$D_{Rf} = D_R$ effect for females
D_{Rmf} = covariance of additive genetic effects in males and females.
Similarly for E_1.

indicating that the genetic effects acting to influence TRC are the same in males and females. We may conclude that the slight heterogeneity observed is due to the inequality in total variance of TRC between males and females (table 1), but that the proportion of total variance which is genetic is the same in both sexes.

Results of fitting models to the data for the separate fingers (table 5) are similar to those for TRC. In all cases environmental models fail badly and, with one exception, one $E_1 D_R$ model gives a good fit to the data and suggests a high heritability for the ridge count on each finger. The exception is the male data for the L2 finger, where there is a particularly strong suggestion of dominance which causes a heterogeneity of fit to the joint data. There is also heterogeneity in the joint data for L4, L5 and R5, where the total variance of females is significantly greater than for males.

The heterogeneity of fit over sexes in these last three variables can be accommodated by fitting the model shown in table 6 to all the twin and sibling data. By fitting the five-parameter model the chi-square for L4 is reduced from $\chi^2_8 = 22{\cdot}35$ to $\chi^2_5 = 6{\cdot}78$ ($\hat{h}^2_f = 0{\cdot}76$, $\hat{h}^2_m = 0{\cdot}84$), for L5 from $\chi^2_8 = 13{\cdot}75$ to $\chi^2_5 = 4{\cdot}46$ ($\hat{h}^2_f = 0{\cdot}92$, $\hat{h}^2_m = 0{\cdot}80$) and for R5 from $\chi^2_8 = 20{\cdot}61$ to $\chi^2_5 = 10{\cdot}0$ ($\hat{h}^2_f = 0{\cdot}82$, $\hat{h}^2_m = 0{\cdot}79$). In each case the heritabilities in males and females are fairly close and the large reductions in chi-square are due mainly to sex differences in total variances (table 1). It is worth noting that Holt (1968; p. 51) found that the female total variances were substantially greater than the male variances for the same three fingers. The biological significance of this is not clear.

In all cases the heritability for the $E_1 D_R$ model in individual counts is lower than for TRC. This indicates negative environmental covariances and for positive genetic covariances between individual counts; this problem will be considered in detail elsewhere (Martin *et al.* 1982 a). In no case does the addition of H_R to the $E_1 D_R$ model cause a significant reduction in chi-square, apart from the anomalous result for L2. There is a high negative correlation between $\hat{D}_R$ and $\hat{H}_R$ because their coefficients in the model are so similar, and this explains the fact that sometimes one of them takes a negative value. Nevertheless, as with TRC, it is noteworthy that large and positive values of $\hat{H}_R$ are obtained for so many fingers.

To see whether detection of genetic non-additivity is sensitive to scale, we minimized skewness by raising the raw measurements to the power 1·5 and did all the calculations again on the transformed data. This had the effect of removing most DZ mean-variance regressions, while not introducing any indication of G × E interaction in MZ twins. The results of model-fitting to the transformed data were very similar to the results for raw data. Chi-square values and heritabilities were hardly altered, and large positive (but non-significant) estimates of H_R were still obtained. This robustness of the partitioning of variance to transformation of the scale of measurement has been noted elsewhere (e.g. Martin and Eysenck 1976).

4. Discussion

The results obtained in this study suggest that genetic variation of finger ridge counts is not determined solely by the simple additive action of genes. Several strands of evidence point to non-additive genetic action with a component in the increasing direction.

Important, although not critical, evidence for this is the negative skewness of the distributions: all fingers show significant negative skewness in at least one sex and so also does the total finger ridge count. Fisher, Immer and Tedin (1932) suggested that the skewness of the distribution may, of itself, be evidence for dominance. Non-additivity of the traits can also be inferred from the negative mean-variance regression

in DZ twins and siblings in the absence of corresponding regressions in MZ twins. Negative skewness and mean-variance regression coefficients may imply the presence of dominant genes acting in the increasing direction. However, it has been shown these could also be produced by unequal gene frequencies in the direction of greater ridge counts (Martin *et al.* 1978), and it is also possible that directional epistasis will have the same effect.

Finally, while mean squares for all the variables except L2 are consistent with a simple additive genetic model, the mere fact that large positive estimates of dominance variance can be estimated, strongly suggests the presence of non-additive genetic variance.

The shape of the distribution of ridge count for an individual finger must in part reflect the distribution of pattern types for the finger, and the distribution of TRC will affect the aggregate of these individual distributions. Any statements we make about genetic non-additivity for ridge counts then, may perhaps be related at a more fundamental biological level to the genetics of pattern type.

A factor which cannot be detected with this data set but which may be confounded with the parameter estimates is an alleged maternal influence on finger ridge counts, particularly on the thumbs (Reed *et al*, 1979). Another difficulty may arise from heterogeneity of MZ twin samples in the extent to which members of pairs shared fetal membranes. Reed *et al.* (1978) found significantly different within-pairs mean sqaures in dichorionic and monochorionic MZ twins for a number of dermatoglyphic characters, including finger ridge counts, but these differences were inconsistent in direction.

The fact that chorion type can influence intra-pair variance in MZ twins suggests that this and other features of the intra-uterine environment may differentially affect intra-pair difference of MZ and DZ twins and siblings. However, since E_1 represents such a small proportion of the total variance, we can be confident that any such biases will not greatly disturb the parameter estimates.

It seems that the present data provide enough evidence to postulate directional non-additivity for ridge counts on invididual fingers and for total ridge count. This is supported by the reanalysis of Holt's family data by Spence *et al.* (1977) and the departure (in the direction of greater counts) of Aboriginal–European hybrids from the mid-parental value (Robson and Parsons 1967, Singh 1979). Our analysis showed that transformation of the data could change the direction of non-addivitiy but not remove it. Further evidence on the directionality of any dominance which may be present could be obtained by counting finger ridges in inbred individuals but we know of no published evidence on this point. Calculation of within-family skewness in sibships of size three or greater may also provide evidence for directional non-additivity (Jinks and Fulker 1970).

The importance of the evidence for directional non-additivity is that it permits inferences about the role of natural selection on the trait. Mather (1973) has argued that a history of directional selection will produce genetic non-additivity in the same direction. In the present case we might infer from the evidence of non-additivity tending to increase finger ridge counts that natural selection has favoured larger counts. It is possible that larger ridge counts confer greater touch sensitivity and this hypothesis is currently being investigated.

In other respects our results confirm earlier findings (summarized by Holt 1968) of high heritability for finger ridge counts and a complete absence of the effects of family environment (E_2). Although these features are broadly similar for all fingers, there

appear to be slight but definite differences in inheritance between fingers, noticeably between the left and right fingers of a pair. Despite the high correlations between finger ridge counts, these differences suggest that there may be genetic effects specific to individual fingers and differing between left and right hands, and this problem will be explored in subsequent papers (Martin *et al.* 1982a, b).

Acknowledgements

The authors wish to thank Dr F. Bielicki and Dr Zofia Świątkowska from the Department of Anthropology of the Polish Academy of Sciences in Wroclaw for providing the prints from twins, which were collected by a team from this Department under project PAN No. II-5; we are also greatly indebted to Dr Danuta Schlesinger from the Institute of Experimental Therapy and Immunology of the Polish Academy of Sciences in Wrocław for performing all the tests related to zygosity determination. Dr Loesch is grateful to the Australian Department of Education for an Australian–Eurpoean Award which enabled the completion of this work at the Australian National University. Dr Loesch's permanent address is Department of Genetics, Psychoneurological Institute, Warsaw, Poland.

References

BOCHEŃSKA, Z., 1964, Inheritance of dermal ridges on the human fingers (in Polish). *Miscellanea, Materialy i Prace Antropologiczne*, **8**, 65, 123–138.

CLARK, P., JARDINE, R., MARTIN, N. G., STARK, A. E., and WALSH, R. J., 1980, Sex differences in the inheritance of some anthropometric characters in twins. *Acta Geneticae Medicae Gemellologiae*, **29**, 171–192.

CLARK, P., STARK, A. E., WALSH, R. J., JARDINE, R., and MARTIN, N. G., 1981a, A twin study of skin reflectance. *Annals of Human Biology*, **8**, 529–541.

CLARK, P., JARDINE, R., JONES, P., MARTIN, N. G., and WALSH, R. J., 1981b, Directional dominance for low IGM and IGA levels. *American Journal of Human Genetics*, **33**, 709–721.

EAVES, L. J., 1977, Inferring the causes of human variation. *Journal of the Royal Statistical Society* A, **140**, 324–355.

EAVES, L. J., and EYSENCK, H. J., 1975, The nature of extraversion: A genetical analysis. *Journal of Personality and Social Psychology*, **32**, 102–112.

FISHER, R. A., IMMER, F. R., and TEDIN, O., 1932, The genetical interpretation of statistics of the third degree in the study of quantitative inheritance. *Genetics*, **17**, 107–124.

HOLT, S. B., 1955, Genetics of dermal ridges: frequency distributions of total finger ridge count. *Annals of Human Genetics, London*, **20**, 159–173.

HOLT, S. B., 1956, Genetics of dermal ridges: parent-child correlations for total finger ridge count. *Annals of Human Genetics, London*, **20**, 270–282.

HOLT, S. B., 1957, Genetics of dermal ridges: sib-pair correlations for total finger ridge count. *Annals of Human Genetics, London*, **21**, 352–367.

HOLT, S. B., 1968, *The Genetics of Dermal Ridges* (Springfield, Ill.: Charles C. Thomas).

JELIESIEJEW, T., and MARCINKIEWICZ, S., 1972, Numbers of cutaneous ridges on the fingers and their heredity in the Polish population. *Folia Morphologica*, **31**, 219–228.

JINKS, J. L., and FULKER, D. W., 1970, Comparison of the biometrical genetical, MAVA and classical approaches to the analysis of human behaviour. *Psychological Bulletin*, **73**, 311–249.

LOESCH, D., 1971, Genetics of dermatoglyphic patterns on palms. *Annals of Human Genetics, London*, **34**, 277–281.

LOESCH, D., 1979, Genetical studies of the palmar and sole patterns and some dermatoglyphic measurements in twins. *Annals of Human Genetics, London*, **43**, 37–53.

MARTIN, N. G., 1975, The inheritance of scholastic abilities in a sample of twins. *Annals of Human Genetics, London*, **39**, 219–229.

MARTIN, N. G., and EYSENCK, H. J., 1976, Genetic factors in sexual behaviour. In *Sex and Personality*, edited by H. J. Eysenck (London: Open Books), Chapter 6.

MARTIN, N. G., EAVES, L. J., KEARSEY, M. J., and DAVIES, P., 1978, The power of the classical twin study. *Heredity*, **40**, 97–116.

MARTIN, N. G., EAVES, L. J., and LOESCH, D. Z., 1982a, A genetical analysis of covariation between finger ridge counts. *Annals of Human Biology* (in the press).

MARTIN, N. G., JINKS, J. L., BERRY, H. S., and LOESCH, D. Z., 1982b, A genetical analysis of diversity and asymmetry in finger ridge counts. *Heredity* (in the press).

MATHER, K., 1973, *Genetical Structure of Populations* (London: Chapman and Hall).
MATHER, K., 1974, Non-allelic interactions in continuous variation of randomly breeding populations. *Heredity*, **32**, 414–417.
MATHER, K., and JINKS, J. L., 1971, *Biometrical Genetics* (London: Chapman and Hall).
MATSUDA, E., and MATSUNAGA, E., 1971, Inheritance of total finger ridge count. *Annual Report of the National Institute of Genetics (Japan)*, **21**, 101–110.
MI, M. P., and RASHAD, M. N., 1975, Genetic parameters of dermal patterns and ridge counts. *Human Heredity*, **25**, 249–262.
REED, T., UCHIDA, I. A., NORTON, J. A., JR., and CHRISTIAN, J. C., 1978, Comparisons of dermatoglyphic patterns in monochorionic and dichorionic monozygotic twins. *American Journal of Human Genetics*, **30**, 383–391.
REED, T., EVANS, M. M., NORTON, J. A., JR., and CHRISTIAN, J. C., 1979, Maternal effects on fingertip dermatoglyphics. *American Journal of Human Genetics*, **31**, 315–329.
ROBSON, M. K., and PARSONS, P. A., 1967, Fingerprint studies in four central Australian Aboriginal tribes. *Archaeology and Physical Anthropology in Oceania*, **II**, 69–73.
SIEMENS, H., 1937, Einige Bemerkungen über die Ähnlichkeitsdiagnose der Eineiigheit. *Archiv für Rassen- und Gesellschaftsbiologie*, **31**, 211–221.
SINGH, S., 1979, Evidence of dominance in the finger ridge counts using multivariate analysis. In *Dermatoglyphics: Fifty Years Later*, edited by W. Wertelecki and C. C. Plato. The National Foundation—March of Dimes, Birth Defects: Original Article Series, vol. 15, number 6. (New York: Alan R. Liss), pp. 495–500.
SPENCE, M. A., WESTLAKE, J., and LANGE, K., 1977, Estimation of the variance components for dermal ridge count. *Annals of Human Genetics, London*, **41**, 111–126.
VERSCHUER, O., 1928, Die Ähnlichkeitsdiagnose der Eineiigkeit von Zwillingen. *Anthropologie*, **5**, 244–258.
VOGELIUS-ANDERSEN, C. H., 1963, On the genetics of certain dermatoglyphic traits. *Proceedings of the 2nd International Congress of Human Genetics* (Rome: Istituto G. Mendel), vol. 3, p. 1509.
WYSLOUCHOWA, B., and ORCZYKOWSKA-ŚWIATKOWSKA, Z., 1969, The diagnosis of twin zygosity on the basis of blood group studies. *Acta Medica Polona*, **10**, 187–198.

Address correspondence to : Dr N. G. Martin, Department of Population Biology, Research School of Biological Sciences, Australian National University, Canberra, Australia.

Zusammenfassung. Eine genetische Analyse der Variation der Fingerleistenzahlen von 221 Zwillingspaaren und 80 Geschwisterpaaren unterschiedlichen Geschlechts wurde durchgeführt. Eine negative Regression der Varianzen der ZZ und Geschwisterpaare auf die Mittelwerte der Paare legt die Wirkung von nicht-additiven Genen oder ungleichmäßigen Genfrequenzen nahe, die die Fingerleistenzahlen erhöhen. Negative Schiefe der Verteilungen unterstützt diese Interpretation. Während Modelle mit Dominanz oder Epistase keine signifikante Verbesserung gegenüber rein additiven genetischen Modellen darstellen, wird es als wichtig angesehen, daß große und positive Werte von nicht additiver genetischer Varianz geschätzt werden. Die evolutionäre Bedeutung von Dominanz und Epistase für größere Fingerleistenzahlen wird diskutiert.

Résumé. Une analyse génétique de la variation des comptes de crêtes digitales de 221 paires de jumeaux et 80 paires de germains de sexe opposé a été menée. Une régression négative des variances des paires de DZ et de germains sur les moyennes des paires suggère l'action de gènes non additifs ou des fréquences géniques inégales tendant à augmenter les comptes de crêtes digitales. L'asymétrie négative des distributions soutient cette vue. Tandis que des modèles incluant la dominance ou l'épistase n'apportent pas d'amélioration significative par rapport à des modèles génetiques purement additifs, il est considéré important que des valeurs élevées et positives de variance génétique non additive sont estimées. L'importance évolutive de la dominance et de l'épistase pour des comptes plus élevés de crêtes digitales est discutée.